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MODERN CONCEPTS OF CARDIOVASCULAR DISEASE

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Auscultation of the Heart: Part I*

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The need for continuing training and education in auscultation of the heart was demonstrated forcefully by the results obtained in testing 523 physicians at the meeting of the American Medical Association in 1959. The test consisted of fifteen tape-recorded "unknowns" which included only commonly heard normal and pathological heart sounds and murmurs. The average number of correct diagnoses was 49 per cent.

It is the purpose of the following discussion to point out some of the basic factors responsible for the production of heart sounds and murmurs, and also some of the factors which limit the ability of the human hearing mechanism to appreciate sounds originating in the thorax.

ORIGIN OF HEART SOUNDS

First Heart Sound

It is now generally agreed that the audible portions of the first and second heart sounds are due to vibrations resulting from closure of the atrioventricular and semilunar valves, respectively. Reference to figure 1 will show that the first heart sound is composed of two components, the mitral and the tricuspid. In late diastole, the left and right atrial pressures are slightly higher than the corresponding ventricular pressures, and the atrioventricular valves are open, allowing blood to flow from atria to ventricles. With the onset of ventricular systole, the pressures in the two ventricles quickly exceed the pressures in the atria, resulting in sudden, forceful closure of the mitral and tricuspid valves. This sudden closure of the valves as the result of abrupt pressure change causes vibrations which, when transmitted through the tissues of the body, are eventually picked up from the precordium by the stethoscope and interpreted as sound by the human hearing mechanism.

Since closure of these valves is often slightly asynchronous, some degree of splitting of the first sound can frequently be detected in normal people, if one listens intently during both phases of respiration. The mitral component of the first sound is usually loudest near the apex, and the tricuspid component may be louder nearer the sternum, so that splitting is sometimes more easily recognized between the sternum and the apex.

The first heart sound is lower in pitch and longer in duration than the second heart sound, possibly because the mitral and tricuspid valves close at low pressure compared with the aortic and pulmonic valves. Because the components of the first sound are lower in frequency and longer in duration, they tend to fuse together, and splitting of the first sound is not as easily recognized as that of the second sound.

The first sound is accentuated in conditions in which the pressure rise within the ventricles is rapid, such as tachycardia resulting from fever, anemia, hyperthyroidism, exercise, or emotional states. It is also accentuated in mitral stenosis.

The first sound is diminished in conditions

* Part II of this article will appear in the April, 1961, issue.

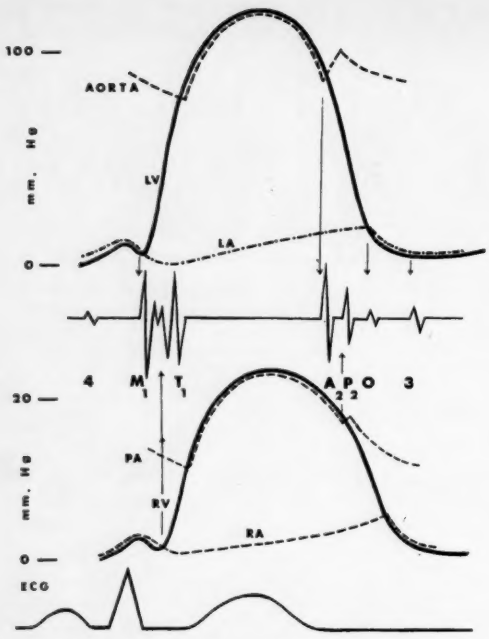

Figure 1. *(LV) = left ventricular pressure curve; (LA) = left atrial pressure curve; (3) and (4) = third and fourth heart sounds; (M_1) = mitral component of the first heart sound; (T_1) = tricuspid component of the first heart sound; (A_2) = aortic component of the second heart sound, (P_2) = pulmonic component of the second heart sound; (O) = normal opening time of the mitral valve; (PA) = pulmonary artery pressure curve; (RV) = right ventricular pressure curve; (RA) = right atrial pressure curve. (Revised from figure reprinted by permission of Grune & Stratton, Inc., from Butterworth, J. S., et al.: Cardiac Auscultation, ed. 2, New York, Grune & Stratton, 1960.)*

in which there is a fall in cardiac output or ventricular contraction is impaired, such as myocardial infarction, shock, constrictive pericarditis, pericardial effusion, cardiac decompensation, and terminal states.

The intensity of the first heart sound may vary from beat to beat in conditions in which there is changing cardiac output, such as atrial fibrillation and atrial flutter with variable block, or when there is dissociation between the atrial and ventricular contractions, such as occurs in complete heart block and ventricular tachycardia.

Second Heart Sound

The second heart sound is initiated by vibrations resulting from the sudden closure of the aortic and pulmonic valves (fig. 1). These valves do not always close at the same time because of the different pressure and flow relationships in the aorta and pulmonary artery. Therefore, splitting of the second sound is common in both normal and pathological circumstances (fig. 2).

Normally the closure of the aortic valve precedes pulmonic valve closure during inspiration, but during expiration closure is synchronous. Asynchronous closure during inspiration is believed to be due to increased filling of the right side of the heart secondary to decreased intrathoracic pressure, with resulting delay in ejection of blood from the right ventricle, and hence a later closure of the pulmonic valve.

The pulmonic component of the second sound is normally heard well only in the pulmonic area and along the left sternal border. It is much fainter than the aortic component, since the aortic valve closes at a pressure some four to six times that of pulmonic valve closure. The aortic component, being louder, is heard well in all areas, and the second sound at the apex is composed entirely of the aortic component.

Rotation of the heart may cause the aortic component of the second sound to be louder in the pulmonic valve area than in the aortic valve area, and this should not be confused with, or arbitrarily called, P_2. It has been customary, in the past, to compare the intensities of the second sound in the aortic and pulmonic areas, but since the aortic component of the second sound is heard well in both areas and may be louder in the pulmonic than in the aortic area, it is more important to identify the two components and to note how the sounds vary with respiration.

The aortic component is usually loud and sharp in systemic hypertension and with dilatation of the ascending aorta. It is diminished or absent in the more severe degrees of aortic stenosis. The pulmonic component is accentuated with conditions resulting in pulmonary hypertension, such as mitral stenosis, left ventricular failure, pulmonary heart disease, primary pulmonary hypertension, and certain congenital lesions. The pulmonic component is diminished with severe pulmonic stenosis.

The splitting of the second sound is increased with conditions which cause delayed pulmonic closure or early aortic closure (fig. 2). In right bundle-branch block, the delayed right-sided conduction results in delayed pulmonic closure, with a resultant wide splitting of the second sound (0.04 to 0.06 seconds). With atrial septal defect, the second sound is

usually widely split and varies relatively little with respiration.

With mitral insufficiency, there may be wider-than-normal splitting due to early closure of the aortic valve, since some of the left ventricular output is regurgitated into the atrium. Another condition in which there is wider-than-normal splitting is mild to moderate pulmonic stenosis. With severe pulmonic stenosis, there is also a delay in the pulmonic component, but the sound is usually too faint to be audible.

Splitting that increases with expiration and decreases with inspiration is called paradoxical splitting. It occurs with conditions in which the aortic component of the second sound is delayed. With left bundle-branch block and aortic stenosis, the aortic closure may occur after the pulmonic; with these conditions, splitting is observed during expiration, when the pulmonic component is early, but is diminished or absent in inspiration, when the pulmonic component is late and hence coincides with the delayed aortic component.

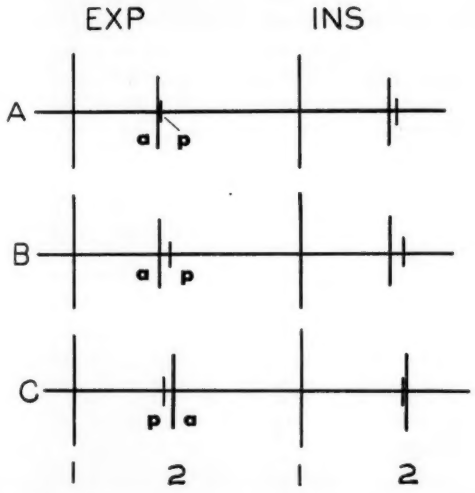

Figure 2. *Schematic diagram of the first and second heart sounds during inspiration and expiration. (A) represents the normal, with fusion of the aortic (a) and pulmonic (p) components of the second sound during expiration and separation (splitting) during inspiration. In this diagram, the aortic component is always represented as louder than the pulmonic component; with increasing pressure in the pulmonary artery, however, the intensity of the pulmonic component (p) may equal or exceed that of the aortic component (a). (B) represents the general group of conditions in which there is separation of the two components during both inspiration and expiration, such as right bundle-branch block. In addition to delay of the pulmonic component, this constant splitting may also be produced by early closure of the aortic valve. (C) represents paradoxical splitting of the second sound, in which fusion occurs during inspiration and splitting is apparent only during expiration. This results from delayed closure of the aortic valve.*

Third Heart Sound

The third heart sound occurs at the end of the period of rapid diastolic filling which follows the opening of the mitral and tricuspid valves (fig. 1). It probably results from the vibrations set up by abrupt limitation of ventricular filling, but tensing of the papillary muscles or valve cusps may also contribute to the sound. The sound, not often heard in normal adults although frequently noted in children, is of low pitch and intensity. It may originate in either the right or the left ventricle. The third sound may be accentuated with any condition in which ventricular dilatation takes place, such as mitral insufficiency, hypertension, and aortic stenosis. It may also be accentuated by an increase in cardiac rate or a failing myocardium. The third heart sound is heard best at the apex, with the subject in a supine position.

Fourth Heart Sound

The fourth heart sound occurs at the time of atrial contraction and is related to the forceful ejection of blood into ventricles that are already distended. It is thus similar in its mechanism to the third heart sound. It is almost always inaudible in normal persons and when heard is of low pitch and intensity. It may be heard near the apex in cases of partial or complete heart block.

SYSTOLIC SOUNDS

Ejection Sounds

Ejection sounds occur at a time when the walls of the aorta or pulmonary artery are distended by the sudden ejection of blood into them. It is possible that the opening of the aortic or pulmonic valve also contributes to the ejection sound and that its mechanism is similar to that of the opening snap of the mitral or tricuspid valve. Whatever the true mechanism, the vibrations set up are not usually audible, but hypertension or dilatation of the aorta or pulmonary artery may accentuate the sounds and render them audible. These sounds are high pitched and are usually heard best at the base, shortly after the first heart sound. At

times, an aortic ejection sound is heard best at the apex, particularly if there is a loud aortic systolic murmur. When the first sound is faint, the ejection sound may easily be mistaken for it. The ejection sound is often misinterpreted as a split first sound. The delay is usually greater than that in a normally split first sound, and when no cause for a widely split first sound is evident an ejection sound should be suspected.

Systolic Clicks

Systolic clicks have a quality similar to that of the ejection sound, but tend to occur in middle, rather than early, systole. There may be some variation with respiration. The resulting three-sound rhythm may be confused with a gallop rhythm, but is usually easily differentiated by the fact that the extra sound is systolic, rather than diastolic, and tachycardia is not present. When there are multiple clicks, they may be misinterpreted as a systolic murmur. Systolic clicks, in themselves, have no known clinical significance, but are thought to arise from the pericardium rather than from within the heart.

DIASTOLIC SOUNDS

Opening Snaps

The opening of the mitral and tricuspid valves occurs when the pressure in the left or right ventricle falls below the pressure in the corresponding atrium. This usually occurs very early in diastole and does not produce an audible sound (see fig. 1). When there is stenosis of the mitral or tricuspid valve, the opening of the diseased valve becomes audible and the sound has a snapping quality. The opening snap is most commonly due to mitral stenosis, since tricuspid stenosis is rare. It is usually heard best along the left sternal border from the second to the fourth intercostal space but may be heard at the apex or, sometimes, over the entire precordium. The opening snap will be discussed further in Part II.

Gallop Rhythm

When the third or fourth heart sound becomes accentuated and the heart rate is rapid, a three-beat rhythm develops which is called a gallop rhythm. In both cases, the extra sound occurs in diastole. When the third heart sound is accentuated, the resulting rhythm is a protodiastolic gallop. When the fourth sound is accentuated, the rhythm is a presystolic gallop. At times both sounds are accentuated, and the rate is so rapid that they merge. This rhythm is known as a summation gallop.

Diastolic Sound in Constrictive Pericarditis

With constrictive pericarditis, a sound is often heard following the second sound, loudest in the region of the xiphoid process. It may be confused with an opening snap, but is generally not so sharp or snapping in quality. This sound may be caused by sudden limitation of ventricular distensibility by a thickened pericardium, and thus is analogous in its mechanism to the physiological third heart sound.

HUMAN HEARING MECHANISM

The human hearing mechanism does not respond equally to all sound frequencies. In general, the lower the frequency (pitch), the greater the intensity (loudness) must be to exceed the threshold of audibility and be recognized as sound. The audible components of heart sounds and murmurs are in the frequency range between 30 and 250 c.p.s. Even the so-called "high-pitched" murmurs of aortic and pulmonic insufficiency are relatively low, falling in the range between 100 and 300 c.p.s. The lower-pitched murmurs of mitral stenosis fall in the range of 30 to 100 c.p.s. Most murmurs are found in the range of 80 to 120 c.p.s., and the normal first and second heart sounds range from 60 to 100 c.p.s. The third and fourth heart sounds, when heard, are usually below 40 c.p.s. Since these and similar sounds and murmurs are frequently barely above the threshold of audibility because of their low frequency and low intensity, and since there is considerable individual variability in the threshold of hearing at any given frequency, it is not difficult to understand why many sounds and murmurs may be heard by one observer and overlooked by another, or by the same observer when conditions for auscultation are not optimal.

A quiet room is most important for careful auscultation. Unfortunately, neither the average office, hospital ward, nor clinic is really quiet.

THE STETHOSCOPE

The stethoscope is another important item in proper auscultation, since the transmission of the sounds of the heart is related to such factors as tubing length, bore, and internal volume. The longer the path from the chest to

the ear, the poorer the transmission of the lower frequencies. Therefore, the tubing should be as short as is consistent with easy use. The ear tips should fit the external auditory canals comfortably but snugly, and the axis of the tubes should be parallel to the axis of the external auditory canals. It is important that the ear tips do not become plugged with wax. The bell chest piece gives maximum low-frequency response, but only when the bell is applied as lightly as possible to the chest wall. With heavy pressure, the skin beneath the bell stretches and acts as a diaphragm. Another point to remember is that the intensity of the transmitted sound is related to the area of contact between the chest piece and the chest wall, and, since the area of the diaphragm is generally greater than that of the open bell, the intensity of sound is usually greater with the former. When the sound is near the threshold of audibility, the increased intensity gained by using the larger area of the diaphragm chest piece may be more important than the low-frequency response of the bell. It is our policy to recommend that each physician select the type of chest piece with which he hears best.

TRANSMISSION THROUGH TISSUES OF THE BODY

A formidable obstacle to auscultation, which may be impossible to overcome except by electronic amplification, is the patient's body. Obesity, thick chest walls, and large breasts may greatly decrease the intensity of heart sounds. Lung tissue is a poor conductor of sound, but blood and muscle are fair conductors, and bone is a good conductor.

ROUTINE OF AUSCULTATION

A routine is essential for thorough auscultatory examination. The sequence of examination is not as important as the development of a consistent habit pattern. The patient should be examined when supine and upright, at rest and after exercise, and during both phases of respiration.

Position

It is important to examine the patient when supine, upright, leaning forward, and in a left lateral position, because some heart sounds and murmurs are heard better in one position than in another.

Respiration

During expiration, the heart is closer to the chest wall, and therefore the heart sounds are louder. Faint murmurs, such as the blowing diastolic murmur of aortic or pulmonic insufficiency, are often heard better during expiration. The effect of respiration on the splitting of the second sound has already been discussed.

Exercise

By increasing the heart rate, and thereby increasing blood flow, both sounds and murmurs may be increased in intensity. This is nonspecific and does not help in differentiating a pathological from a nonpathological murmur, but it may be useful in intensifying a murmur or sound which is just at the threshold of audibility.

Timing

The first and second heart sounds should be identified in each area. Generally, the second sound is loudest at the base and, from there, can be gradually traced to other areas by small increments of movement of the stethoscope. At the apex, the first sound is usually louder, longer in duration, and lower in pitch than the second sound. At heart rates below 100 per minute, the diastolic interval is longer than systole, and therefore the sound occurring after the longer pause is the first heart sound. Another method of identifying the sounds is by simultaneous palpation of the carotid pulse, the first sound being approximately synchronous with the thrust of the carotid pulse. After identifying the first and second sounds and evaluating their relative intensities, the examiner should then concentrate on the systolic and diastolic phases to detect additional sounds and murmurs, if present. The careful physician will often re-examine areas several times to be sure of his observations.

While there are classical valve areas where the physician is accustomed to listen, it should be emphasized that the sounds and murmurs originating from a valve are not always heard best in that particular valve area, and thus it is important to examine by auscultation the entire precordium.

REFERENCES

All references for this article will appear at the end of Part II in the April, 1961, issue.

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Bal Harbour, Miami Beach, Florida

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Council on Arteriosclerosis-American Society for the Study of Arteriosclerosis

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